3. (**Amended**) A method for treating a disorder characterized by neuronal cell loss, comprising administering to a mammal a composition comprising a molecule that overcomes morphogen inhibition, thereby to potentiate growth-promoting effects of endogenous morphogens.

4. (**Amended**) A method for treating a neurodegenerative disorder, comprising administering to a mammal a composition comprising a molecule that overcomes morphogen inhibition.

5. (**Reiterated**) The method of claim 1, wherein said morphogen activity is endogenous.

6. (**Reiterated**) The method of claim 1, wherein said morphogen activity is the result of an exogenously provided morphogen.

7. (**Reiterated**) The method of claim 4, wherein said composition further comprises a morphogen.

8. (**Amended**) The method of claim 3 or 4, wherein said disorder is Alzheimer's disease, Parkinson's disease, Huntington's disease, senile dementia, alcohol-induced dementia, or stroke.

9. (**Amended**) The method of claim 1, 2, 3 or 4, wherein said agent that overcomes morphogen inhibition is a cytokine antagonist, a retinoid antagonist, or a protein kinase A inhibitor.

10. (**Reiterated**) The method of claim 9, wherein said cytokine antagonist is a neuropoetic cytokine antagonist.

11. (**Amended**) The method of claim 10, wherein said neuropoetic cytokine antagonist is an LIF antagonist or a CTNF antagonist.

12. (**Amended**) The method of claim 11, wherein said LIF antagonist is a monoclonal antibody to the gp130 protein.

13. (**Reiterated**) The method of claim 9, wherein said retinoid antagonist is a retinoic acid receptor antagonist.

14. (**Reiterated**) The method of claim 9, wherein said retinoid antagonist is a retinoid X receptor antagonist.

15. (**Amended**) The method of claim 9, wherein said protein kinase A inhibitor is (2-p-bromocinnamylaminoethyl)-isoquinolinesulfonamide, an enantiomer of dibutyryl cAMP, or an enantiomer of cAMP.

16. (**Amended**) The method of claim 7, wherein said morphogen comprises an amino acid sequence selected from a sequence: (a) having at least 70% homology with the C-terminal seven-cysteine skeleton of human OP-1, residues 330-431 of SEQ ID NO: 2; (b) having greater than 60% amino acid sequence identity with said C-terminal seven-cysteine skeleton of human OP-1; (c) defined by Generic Sequence 7, SEQ ID NO: 4; (d) defined by Generic Sequence 8, SEQ ID NO: 5; (e) defined by Generic Sequence 9, SEQ ID NO: 6; (f) defined by Generic Sequence 10, SEQ ID NO: 7; or (g) defined by OPX, SEQ ID NO: 3.

17. (**Amended**) The method of claim 7, wherein said morphogen is human OP-1, mouse OP-1, human OP-2, mouse OP-2, 60A, GDF-1, BNT2A, BMT2B, DPP, Vgl, Vgr-1, BNW3, BNW5, or BW6.

18. (**Reiterated**) The method of claim 7, wherein said morphogen is OP-1.

19. (**Amended**) The method of claim 1, wherein the molecule binds an endogenous ligand for a cytokine receptor or a retinoid receptor.

20. (**Reiterated**) The method of claim 19, wherein said cytokine receptor is a neuropoetic cytokine receptor.

21. (**Amended**) The method of claim 20, wherein said neuropoetic cytokine receptor is an LIF receptor or a CTNF receptor.

22. (**Reiterated**) The method of claim 19, wherein said retinoid receptor is a retinoic acid receptor.

23. (**Reiterated**) The method of claim 19, wherein said retinoid receptor is a retinoid X receptor.

24. (**Amended**) The method of claim 1, wherein the molecule is a cAMP-dependent messenger pathway inhibitor.

25. (**Amended**) The method of claim 24, wherein said cAMP-dependent messenger pathway inhibitor comprises a protein kinase A inhibitor.

26. (**Amended**) The method of claim 25, wherein said protein kinase A inhibitor is (2-p-bromocinnamylaminoethyl)-isoquinolinesulfonamide, an enantiomer of dibutyryl cAMP, or an enantiomer of cAMP.

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27. (**Amended**) A screening method for identifying a molecule that potentiates morphogen activity, comprising (1) providing a test cell comprising a morphogen inhibitory element, wherein said test cell, when contacted with OP-1, does not undergo tissue morphogenesis; (2) exposing said test cell to OP-1 and a candidate molecule; and (3) identifying a molecule that potentiates morphogen activity as a candidate that overcomes morphogen inhibition and permits said test cell to undergo OP-1-induced tissue morphogenesis.

28. (**Amended**) The screening method of claim 27, wherein said test cell is obtained from: sympathetic nerves, hippocampus, cerebral cortex, striatum, kidney, liver, adrenals, urinary bladder, or testes.

29. (**Reiterated**) A molecule identified by the method of claim 27.

30. (**Reiterated**) The molecule of claim 29, wherein said molecule is a protein.

31. (**Reiterated**) The molecule of claim 29, wherein said molecule is an inorganic molecule.

32. (**Reiterated**) The molecule of claim 29, wherein said molecule is an organic molecule.